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L3	lymphotoxin and shock	935	L3
L2	L1 and lymphotoxin	9	L2
L1	browning J.in.	130	L1

END OF SEARCH HISTORY

1: Cancer Biother Radiopharm 1998 Jun;13(3):193-207 Related Articles, Links

Implications of the analogy between recombinant cytokine toxicities and manifestations of hantavirus infections.

Wimer BM.

JBMW Immunotherapeutics, Albuquerque, NM 87123-4255, USA.

The etiologic hantavirus of the 1993 emergence of an acute pulmonary failure syndrome in the area around northwestern New Mexico was quickly recognized as related to the Hantaan virus responsible for the outbreak of Korean epidemic hemorrhagic fever (EHF) among UN troops in 1951. Discovery of the new disease which was named the hantavirus pulmonary syndrome (HPS) and its causative agent the Sine Nombre virus (SNV) inspired detailed comparisons between the two disorders. Major damage to the epithelial cells of the capillaries and arterioles throughout the body leading to extensive capillary leak and subsequent hypotension and shock was the common denominator. The lung capillaries and arterioles were the focus of attack that could lead to rapid pulmonary failure in HPS and the corresponding renal and retroperitoneal vessels that caused a more protracted illness in EHF, but both displayed remarkably similar peripheral blood abnormalities including abnormal mononuclear cells, immature neutrophilia, thrombocytopenia, and hemoconcentration characteristic enough to make blood smear examination a useful tool in early diagnosis. There are evidences that a heavy virus presence in the involved endothelial cells is accompanied by various mononuclear cells capable of generating potent immune response in these areas. Relevant toxic effects of systemically-administered high-dose interleukin-2 for resistant cancers include fever, chills, diarrhea, renal dysfunction, capillary leak syndrome accompanied by hypotension requiring aggressive pressor support, and occasional pleural effusions with diffuse pulmonary infiltrates and hypoxia severe enough to require ventilatory assistance. Peripheral blood mononuclear cells cultured in vitro with IL-2 secrete secondary cytokines such as IL-1, TNF-alpha, and interferon-gamma (IFN-gamma). TNF-alpha, implicated in the pathophysiology of septic shock, is capable of inducing adult respiratory distress syndrome (ARDS) in experimental animals and humans. The strong similarity of these effects to the manifestations noted in the hantavirus diseases justifies the conviction that these and other cytokines involved in potent immune responses would constitute the pathogenic toxic substances predicted by perceptive early investigators of EHF. This concept is favored by clear indications that in both diseases active virus infection disappears the first few days and the ages of involvement correlate with periods of immunocompetence. The paradox of

systemic injections of IL-2 that risk hantavirus-type toxicities for treating renal cell carcinoma and melanoma might be avoided by giving potentially more efficacious plant mitogens like PHA as previously reported. The expanded disclosure of a collaborator's method suggesting superior potential for cancer cure involves a unique application of pokeweed mitogen that delivers various cellular and cytokine responses directly to the tumor.

Publication Types:
- Review
- Review, Tutorial

PMID: 10850356 [PubMed - indexed for MEDLINE]

1: Exp Lung Res 1993 Nov-Dec;19(6):715-29 Related Articles, Links

Pichinde virus-induced respiratory failure due to obstruction of the small airways: structure and function.

Schaeffer RC Jr, Bitrick MS Jr, Connolly B, Jenson AB, Gong F.

Department of Physiology, University of Arizona Health Sciences Center, Tucson 85724.

Respiratory distress that leads to death is seen in patients with Lassa fever. The development of this respiratory problem was studied using a Pichinde virus model (10(4) plaque forming units, IP, survival time 20 +/- 1 days) in strain 13 guinea pigs (n = 35, 229-353 g) of this lethal human contagious infectious disease. Extravascular lung water to bloodless dry lung weight (EVLW/BDLW) ratio showed a modest yet significant increase in animals 13 and 18-21 days postinoculation (PI). In contrast, residual lung blood and lung radioactive 125I-labeled human serum albumin activity index were elevated only in the 18- to 21-day group. These data are consistent with the progressive severity of perivascular edema, lymphocytic pneumonitis, and some alveolar protein between days 13 and 18-21 PI. Lymphocytic pneumonitis appeared to be distributed near most airways and was proportional to the degree of Pichinde virus antigen staining of alveolar macrophages, large mononuclear cells within the pulmonary vascular and extravascular spaces, and alveolar-capillary membranes. These findings suggest that lymphocyte recruitment to the lung reflects the Pichinde virus-induced cell-mediated immune response. Obstructed small bronchi with some lumenal cell debris and hypertrophied epithelial cells were found associated with the areas of marked pneumonitis. The severe hypoxemia and modest anaerobic metabolism in association with marked tachypnea and normocapnia are consistent with small airway obstruction and wasted ventilation, since no change in arterial blood pressure, heart rate, hematocrit, hemoglobin, or blood volume was noted. These data suggest that Pichinde virus-induced respiratory failure was due to obstruction of the small airways with wasted ventilation in association with lymphocytic pneumonitis.

PMID: 8281916 [PubMed - indexed for MEDLINE]

1: Indian J Chest Dis Allied Sci 1999 Apr-Jun;41(2):115-9 Related Articles, Links

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Dengue hemorrhagic fever (DHF) presenting with ARDS.

Sen MK, Ojha UC, Chakrabarti S, Suri JC.

Department of Pulmonary, Critical Care and Sleep Medicine, Safdarjung Hospital, New Delhi.

A patient of dengue hemorrhagic fever who progressed into acute respiratory distress syndrome (ARDS) is reported. The patient recovered after mechanical ventilation and supportive treatment.

PMID: 10437325 [PubMed - indexed for MEDLINE]

1: Rev Infect Dis 1983 Mar-Apr;5(2):346-52 Related Articles, Links

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Delayed-type hypersensitivity: probable role in the pathogenesis of dengue hemorrhagic fever/dengue shock syndrome.

Pang T.

The hypothesis presented proposes the involvement of a systemic form of a delayed-type hypersensitivity reaction in the pathogenesis of dengue hemorrhagic fever/dengue shock syndrome. It envisages the activation of sensitized T lymphocytes during a secondary infection by viral antigen present on the surfaces of mononuclear phagocytic cells. These antigen-activated T cells then release a variety of biologically active chemical mediators (lymphokines), which then produce the symptoms of shock and hemorrhage seen in cases of dengue hemorrhagic fever/dengue shock syndrome.

PMID: 6844807 [PubMed - indexed for MEDLINE]